1. A chimeric live, infectious, attenuated virus, comprising:

a yellow fever virus in which the nucleotide sequence encoding a prM-E protein is either deleted, truncated, or mutated so that functional yellow fever virus prM-E protein is not expressed, and

integrated into the genome of said yellow fever virus, a nucleotide sequence encoding a prM-E protein of a second, different flavivirus, so that said prM-E protein of said second flavivirus is expressed.

2. The chimeric virus of claim 1, wherein said second flavivirus is a Japanese Encephalitis (JE) virus.

3. The chimeric virus of claim 1, wherein said second flavivirus is a Dengue virus selected from the group consisting of Dengue types 1-4.

4. The chimeric virus of claim 3, wherein said nucleotide sequences derived from said Dengue virus are derived from two or more different Dengue strains.

5. The chimeric virus of claim 1, wherein said second flavivirus is selected from the group consisting of a Murray Valley Encephalitis virus, a St. Louis Encephalitis virus, a West Nile virus, a Tick-borne Encephalitis virus (*i.e.*, a Central European Encephalitis virus or a Russian Spring-Summer Encephalitis virus), a Hepatitis C virus, a Kunjin virus, a Powassan virus, a Kyasanur Forest Disease virus, and an Omsk Hemorrhagic Fever virus.

6. The chimeric virus of claim 1, wherein the nucleotide sequence encoding the prM-E protein of said second, different flavivirus replaces the nucleotide sequence encoding the prM-E protein of said yellow fever virus.

7. The chimeric virus of claim 1, wherein said nucleotide sequence encoding said prM-E protein of said second, different flavivirus comprises a mutation that prevents prM cleavage to produce M protein.

8. The chimeric virus of claim 1, wherein the NS2B-3 protease recognition site and the signal sequences and cleavage sites at the C/prM and E/NS1 junctions are maintained in construction of said chimeric flavivirus.

9. A method of preventing or treating flavivirus infection in a patient, said method comprising administering to said patient a chimeric, live, infectious, attenuated virus comprising:

a yellow fever virus in which the nucleotide sequence encoding a prM-E protein is either deleted, truncated, or mutated so that functional yellow fever virus prM-E protein is not expressed, and

integrated into the genome of said yellow fever virus, a nucleotide sequence encoding a prM-E protein of a second, different flavivirus, so that said prM-E protein of said second flavivirus is expressed.

10. The method of claim 9, wherein said second flavivirus is a Japanese Encephalitis (JE) virus.

11. The method of claim 9, wherein said second flavivirus is a Dengue virus selected from the group consisting of Dengue types 1-4.

12. The method of claim 11, wherein said nucleotide sequences derived from said Dengue virus are derived from two or more different Dengue strains.

13. The method of claim 10, wherein said second flavivirus is selected from the group consisting of a Murray Valley Encephalitis virus, a St. Louis Encephalitis virus, a West Nile virus, a Tick-borne Encephalitis virus, a Hepatitis C virus, a Kunjin virus, a Central European Encephalitis virus, a Russian Spring-Summer Encephalitis virus, a Powassan virus, a Kyasanur Forest Disease virus, and an Omsk Hemorrhagic Fever virus.

14. The method of claim 10, wherein the nucleotide sequence encoding the prM-E protein of said second, different flavivirus replaces the nucleotide sequence encoding the prM-E protein of said yellow fever virus.

15. The method of claim 10, wherein said nucleotide sequence encoding said prM-E protein of said second, different flavivirus comprises a mutation that prevents prM cleavage to produce M protein.

16. The method of claim 10, wherein the NS2B-3 protease recognition site and the signal sequences and cleavage sites at the C/prM and E/NS1 junctions are maintained in construction of said chimeric flavivirus.

17. A nucleic acid molecule encoding a chimeric live, infectious, attenuated virus comprising:

a yellow fever virus in which the nucleotide sequence encoding a prM-E protein is either deleted, truncated, or mutated so that functional yellow fever virus prM-E protein is not expressed, and

integrated into the genome of said yellow fever virus, a nucleotide sequence encoding a prM-E protein of a second, different flavivirus, so that said prM-E protein of said second flavivirus is expressed.

18. The nucleic acid molecule of claim 17, wherein said second flavivirus is a Japanese Encephalitis (JE) virus.

19. The nucleic acid molecule of claim 17, wherein said second flavivirus is a Dengue virus selected from the group consisting of Dengue types 1-4.

20. The nucleic acid molecule of claim 19, wherein said nucleotide sequences derived from said Dengue virus are derived from two or more different Dengue strains.

21. The nucleic molecule of claim 17, wherein said second flavivirus is selected from the group consisting of a Murray Valley Encephalitis virus, a St. Louis Encephalitis virus, a West Nile virus, a Tick-borne Encephalitis virus (*i.e.*, a Central European Encephalitis virus or a Russian Spring-Summer Encephalitis virus), a Hepatitis C virus, a Kunjin virus, a Powassan virus, a Kyasanur Forest Disease virus, and an Omsk Hemorrhagic Fever virus.

22. The nucleic acid molecule of claim 17, wherein the nucleotide sequence encoding the prM-E protein of said second, different flavivirus replaces the nucleotide sequence encoding the prM-E protein of said yellow fever virus.

23. The nucleic acid molecule of claim 17, wherein said nucleotide sequence encoding said prM-E protein of said second, different flavivirus comprises a mutation that prevents prM cleavage to produce M protein.

24. The nucleic acid molecule of claim 17, wherein NS2B-3 protease recognition site and the signal sequences and cleavage sites at the C/prM and E/NS1 junctions are maintained in construction of said chimeric flavivirus.

25. A method of producing a gene product in a cell in a patient, said method comprising introducing into said cell a yellow fever virus vector comprising a gene encoding said gene product.

26. The method of claim 25, wherein said cell is a cell of the lymphoid system or the reticuloendothelial system, or a precursor thereof.

27. The method of claim 25, wherein said patient has cancer.

28. The method of claim 27, wherein said cancer is leukemia.

29. The method of claim 27, wherein said gene product is a tumor antigen or a cytokine.